Blood Lead Levels among High-Risk Children, Detroit, Michigan

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Abstract: Since 1972, a federally funded lead screening program has been operational in Detroit, Michigan. Blood lead screening data (27,430 initial tests on Black children ≤6 years) were assigned by census tract of child's residence to one of the 15 socioeconomically similar areas in geographic proximity to one another. Higher blood lead levels were associated with lower education, lower income, and increased proportion of single parent families. This emphasizes the need to continue screening efforts in lower socioeconomic areas with older housing. (*Am J Public Health* 1982; 72:1288–1290.)

Introduction

Considerable public funds have been spent in the past decade on childhood lead screening programs, and large data sets describing the results have been accumulated. More recently, subsets of these data have been compiled and analyzed for various cities. Socioeconomic factors and exposure indices have been related to observed blood lead levels over time.[1-3*]

The federally funded Detroit, Michigan program became operational in 1972. Since its inception, over 60,000 children have been screened. Of the estimated 100,967 Black children (≤6 years of age) in Detroit, approximately 37,000 have received at least one blood lead screening test.

Quantification of blood lead levels was undertaken using the dithizone extraction colormetric method[4] from January 1972 through December 1973 and Atomic Absorption Spectrophotometry[5] from August 1973 until December 1978. The accuracy and precision of the two methods were similar.

The entire city of Detroit was "targeted" for screening. Sixty per cent of the city's housing units were considered to be in the "lead belt," i.e., the area containing dilapidated and deteriorated housing. This area was given top priority for screening efforts. Because of the overwhelming number of Black children that were screened (90 per cent of the total study population), we have limited analysis to Black children ≤6 years of age for whom race-specific information was available by census tract of residence (N=27,430). The purpose of this investigation is to study the association between socioeconomic indicators and blood lead levels in Detroit Black children ≤6 years of age.

Methods and Materials

The 1970 US Census Data[6] for Detroit was obtained and a summary of salient demographic variables for each of the 447 Detroit census tracts was evaluated. The factors chosen for study include: 1) per cent of dwellings built prior to 1950; 2) median family income; 3) mean male education; 4) mean female education; 5) per cent of families with a female head and children ≤6 years; and 6) per cent females separated or divorced. Mean values for the total Black population of each census tract were then grouped into quintiles for the city and coded to aid in clustering. Race-specific census information was available on only 270 of the 447 census tracts.

Census tracts that were sociodemographically similar and in geographic proximity to one another were then combined to form 15 contiguous areas. Natural neighborhood boundaries were also given consideration in determining the Detroit clusters. A sufficient number of children were screened in each area to permit comparisons.

An analysis of variance on each factor demonstrated a significant difference between areas. To assure within-group homogeneity, the mean of each socioeconomic variable by census tract was compared with its mean area. The acceptance criteria was that the mean for the census tract lay within one standard deviation of the area mean. There were very few discrepancies. Those census tracts which fit another nearby area more clearly were reassigned. Table 1 shows the distribution of various race-specific sociodemographic variables for the 15 Detroit areas.

Results

In the evaluation of individual blood lead levels, it became apparent that blood lead levels demonstrated a log

*Ter Haar G, Chadzynski L: An Investigation of Elevated Blood Lead Levels in Detroit Children. (Pre-publication draft) April 11, 1977.

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TABLE 1—Distribution of Sociodemographic Variables and Geometric Mean Blood Lead Levels for Blacks, 15 Detroit Areas

Area	X̄ Male Education	X̄ Female Education	X̄ % Pre-1950 Housing	X̄ Family Income	X̄ % Female Divorced/ Separated	X̄ % Families with Female Head, Children <6 Years	X̄ Geometric Blood Lead
1	11.08	11.34	62.98	151.88	8.97	1.96	20.96
2	10.93	10.80	72.00	119.07	13.53	8.07	22.59
3	11.18	11.68	72.37	161.47	12.09	4.55	22.00
4	9.93	10.45	85.02	108.27	13.74	11.16	24.36
5	9.81	10.32	92.86	104.17	16.50	9.59	24.63
6	9.63	10.07	95.40	97.00	18.03	16.05	25.70
7	8.55	9.36	97.00	80.76	18.50	18.03	28.02
8	8.36	8.85	98.62	83.63	13.09	22.40	26.95
9	9.62	10.33	72.54	107.83	15.36	13.47	24.96
10	7.99	8.76	97.70	77.22	19.71	20.33	29.36
11	8.51	9.23	88.54	83.91	17.75	21.47	26.83
12	8.34	8.73	94.51	72.44	20.65	20.26	28.24
13	9.34	9.71	91.84	75.17	18.54	17.65	27.89
14	8.55	9.16	92.60	86.58	17.16	16.35	27.55
15	9.18	9.63	92.65	91.85	20.63	23.71	27.06
TOTAL	9.3595	9.8517	88.7581	98.5203	16.7250	15.4426	26.23
	±0.9753	±0.8369	±11.3654	±25.1206	± 9.0122	±13.3434	
	N = 273 cts	N = 270 cts	N = 223 cts	N = 271 cts	N = 272 cts	N = 272 cts	
Correlation with geometric Mean Blood Lead	r = −.949	r = −.9405	r = .871	r = −.9401	r = +.854	r = +.905	

15 areas, 272 census tracts

normal distribution. Therefore, the geometric mean is more appropriate than the arithmetic mean for analysis. The blood lead distribution varies markedly with a geometric mean level of 20.96 μg/dl in area 1 to a high value of 29.3 μg/dl in area 10 (mean 26.33).

Mean male and female education and median income were all highly intercorrelated with each other and inversely correlated with the geometric mean blood lead level. This observation has been noted previously.[7-9] Areas with a high percentage of families with a female head and with children ≤6 years and those with a greater than average per cent of females who were divorced or separated also reported higher mean blood lead levels.

Pearson's product moment correlation coefficients were determined (bottom of Table 1). Mean male and female education and median family income were highly negatively correlated with geometric mean blood lead level. As might be expected, those variables associated with single parent families (per cent females separated or divorced and per cent females head of household with young children) were highly positively correlated with increased blood lead levels in children.

Multiple regression analysis of the independent variables was carried out to assess the relation to the dependent variable, geometric mean blood lead level, with concomitant adjustment for the effect of other variables. The coefficient of determination (R^2) was 95.8 per cent, indicating that a substantial amount of the variation was accounted for by our model. The overall regression was significant at $p < 0.005$. Mean male and female education explained 90 per cent of the variance in blood lead levels and was significant at $p < 0.005$. Per cent females divorced or separated explained an additional 5.8 per cent of the variation and was significant at $p < 0.025$. The high intercorrelation of all the independent variables substantially restricts the multiple regression analysis.

Discussion

An important consideration for all research of this type pertains to the validity of using census data to describe our study population. We must assume that the screened population is representative of the tract from which it came, an assumption which may or may not be valid.

Historically, the primary source of lead exposure to children has been considered to be lead-based paint in old housing. However, in Detroit, an area with a uniformly high percentage of old housing, screened children exhibited a marked variation in blood lead levels. This would appear to indicate that although a lead source may exist, the actual exposure to lead and resultant illness may be very strongly influenced by the sociodemographic characteristics of the population at risk. Those with greater financial resources may be more able to plan child activities away from their home, afford more supportive day care, etc.

As noted earlier, there is a high percentage of single parent families with small children in the Detroit area who are headed by females. According to the US Bureau of the Census in 1978, children of families headed by women were six times more likely to be below the poverty level. The income differential between Black and White people, coupled with the high proportion of Black children living with mothers only, accounts for a much higher proportion of

Black than White children who live in poverty (41 per cent versus 11 per cent in 1978).[10]

Our finding of higher blood lead levels associated with lower education, lower income, and increased proportion of single parent families emphasizes the need to continue screening efforts in the lower socioeconomic areas with older housing. Further research into non-traditional sources of lead exposure and intervening factors is indicated.

REFERENCES

1. Public Law 91-695, January 13, 1971, 91st Congress, HR 19172.
2. Enterline PE, Waller J, Henderson *et al*: Final Report on Planning Grant to Evaluate Blood Lead Data for Children. Washington, DC: National Bureau of Standards 6-9004, 1977.
3. Stark AD, Meigs JW, Fitch RA: Family operational co-factors in the epidemiology of childhood lead poisoning. Arch Environ Health Sept/Oct 1978; 33:222–225.
4. Keenan RG, *et al*: USPHS method for determining lead in air and biological materials. Am J Ind Hyg 1963; 24:481–91.
5. Hessel DW: A simple and quantitive determination of lead in blood. Atomic Absorption Newsletter 1968; 7:55.
6. US Bureau of Census: Detailed Demographic Characteristics of Detroit SMSA Census Tracts, 1970. Washington, DC: Census Bureau, 1970.
7. Ingalls TH, Tiboni EA, Werrin M: Lead poisoning in Philadelphia. Arch Environ Health 1961; 3:575–579.
8. Jacobziner H: Lead poisoning in childhood: epidemiology, manifestations and prevention. Clin Pediatrics 1966; 5:277–286.
9. Christian JR, Celewyzc BS, Andelman SL: A three-year-study of lead poisoning in Chicago. Am J Public Health 1964; 54:1241–1251.
10. Report of the Select Panel for the Promotion of Child Health; 1980: Better Health for our Children: A National Strategy. III. A Statistical Profile. Washington, DC: US Department of Health and Human Services.

A Brief Review of the Current Status of Alternatives to Chlorine Disinfection of Water

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Abstract: This paper briefly outlines some of the alternative disinfectants being considered in lieu of chlorination. Methods currently in use as well as those in the research stage are included. Each method is assessed with respect to disinfection efficiency and environmental impact. (*Am J Public Health* 1982; 72:1290–1293.)

Introduction

Chlorine is currently being reevaluated as the standard for disinfection of drinking water and wastewater. Alternative methods are being sought due to the cost of manufacture of hypochlorite, its potential carcinogenic effects,[1] mutagenic effects,[2] toxicity to aquatic species,[3,4] and explosive properties.[5] Among the most promising chemical alternatives are chlorine dioxide and ozone.

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Chlorine Dioxide

Approximately 85 water treatment plants in the United States currently use chlorine dioxide for disinfection, and for removing iron, manganese, taste, odor and color. In Europe, approximately 495 plants use the compound for disinfection and as an oxidant residual.[6]

Chlorine dioxide effectively destroys coliforms, enteroviruses,[7] and pathogenic amoebae.[4] It is a stronger oxidant than chlorine and also provides a longer residual in potable water. When chlorine is absent from water, chlorine dioxide does not react with ammonia or aromatic organics and does not produce trihalomethanes.[9] It is also less likely than chlorine to form chlorinated organics.[6] Disadvantages of chlorine dioxide include its cost and production problems. In the generation of chlorine dioxide, free chlorine, chloramines, and traces of chlorite and chlorate are produced. Ammonia is generally added to the feed water to combine with free chlorine to produce chloramines and prevent the formation of trihalomethanes. However, if excess chlorine is present, trihalomethanes are formed.[9] Both chlorite and chlorate can oxidize hemoglobin resulting in methemoglobin and reduced oxygen carrying capacity. Chlorite is a hemolytic agent and may initiate hemolytic anemia in susceptible individuals at the levels found following disinfection.[10]

Ozone

Since the first ozonation plant was constructed in 1893, over 1,000 plants have been built throughout the world.[6] In